Exhibit 27

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Specific Causation Expert Report: Frank Mousser

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I use these charts to add weight to the differential diagnosis analyses below.

VIII. Levels of the Toxins Known to Cause Kidney Cancer:

I have reviewed the general causation reports of Drs. Hatten and Bird. These experts go through a detailed analysis of the epidemiologic and toxicologic literature and science, as well as the mechanism of injury for the different toxins. They also detail the levels at which each of these toxins is hazardous to humans generally and that are known to cause kidney cancer. For example, the following levels were discussed in these reports:

1. **Cumulative exposure to 27-44 mg of PCE**: Aschengrau A, Ozonoff D, Paulu C, et al. Cancer risk and tetrachloroethylene-contaminated drinking water in Massachusetts. *Arch Environ Health*. 1993;48(5):284-292.

2. **Exposure to a TCE concentration of > 76 ppb**: Moore LE, Boffetta P, Karami S, et al. Occupational trichloroethylene exposure and renal carcinoma risk: evidence of genetic susceptibility by reductive metabolism gene variants. *Cancer Res*. 2010;70(16):6527-6536.

3. **Cumulative exposure of > 1,580 ppb-years**: Moore et al., 2010.

4. **Sustained exposure to 0-25 ppb of TCE**: Andrew AS, Li M, Shi X, Rees JR, Craver KM, Petali JM. Kidney Cancer Risk Associated with Historic Groundwater Trichloroethylene Contamination. *Int J Environ Res Public Health*. 2022;19(2):618.

5. **Exposure to a TCE concentration of 267 ppb**: Parker GS, Rosen, SL. Woburn: Cancer Incidence and Environmental Hazards 1969-1978. Commonwealth of Massachusetts, Department of Public Health, 1981.

6. **Exposure to a PCE concentration of 21 ppb**: Parker and Rosen, 1981.

7. **Cumulative exposure of 1 - 3,100 µg/L-month of TCE**: Bove FJ, Ruckart PZ, Maslia M, Larson TC. Evaluation of mortality among Marines and Navy personnel exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. *Environ Health*. 2014;13:10.

8. **Cumulative exposure of 1 - 155 µg/L-month of PCE**: Bove et al, 2014a.

9. **Cumulative exposure of 1 – 4,600 µg/L-month of exposure to all compounds at Camp Lejeune**: Bove et al, 2014a.

10. **Cumulative exposure of 3,100 – 7,700 µg/L-month of TCE**: Bove et al, 2014a.

11. **Cumulative exposure of 155 - 380 µg/L-month of PCE**: Bove et al, 2014a.

12. **Cumulative exposure of 4,600 – 12,250 µg/L-month of exposure to all compounds at Camp Lejeune**: Bove et al, 2014a.

13. **Cumulative exposure greater than 7,700 µg/L-month of TCE**: Bove et al, 2014a.

14. **Cumulative exposure greater than 380 µg/L-month of PCE**: Bove et al, 2014a.

15. **Cumulative exposure greater than 12,250 µg/L-month of exposure to all compounds at Camp Lejeune**: Bove et al, 2014a.

16. **18 months of residence on base from 1975 to 1985**: Bove et al, 2014a.

17. **Employment on base for 2.5 years**: Bove FJ, Ruckart PZ, Maslia M, Larson TC. Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. *Environ Health*. 2014;13:68.

18. **Cumulative exposure to 110 – 11,030 ppb-months of TCE**: Agency for Toxic Substances and Disease Registry (ATSDR). *Morbidity Study of Former Marines, Employees, and Dependents Potentially Exposed to Contaminated Drinking Water at U.S. Marine Corps Base Camp Lejeune*. April 2018.

19. **Cumulative exposure to 36 - 711 ppb-months of PCE**: ATSDR, 2018.

20. **Cumulative exposure greater than 11,030 ppb-months of TCE**: ATSDR, 2018.

21. **Cumulative exposure greater than 711 ppb-months of PCE**: ATSDR, 2018.

22. **1-6 quarters stationed on base as a service member from 1975 to 1985**: Bove FJ, Greek A, et al. Cancer Incidence among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water at US Marine Corps Base Camp Lejeune: A Cohort Study. *Environ Health Perspect* 2024b;132;10.

23. **More than 21 quarters spent on base as a civilian worker from 1975 to 1985**: Bove et al, 2024b.

Further, in the discussion of the section detailing levels that are hazardous to humans and that are known to cause kidney cancer, the following was stated in terms of the strength of the science:

> Determination of the levels of exposure that are hazardous to humans, and known to cause kidney cancer, follows a framework of evidence. The most relevant literature provides estimates aligned with the population and exposure of concern. Accordingly, if these publications are sufficient to inform the question of exposure levels associated with the outcome of interest, there is no need to turn to alternative exposure metrics from the greater body of literature. Of note, unless specific subgroup analyses of vulnerable populations occur, then reported levels of exposures are likely to be overestimated for such individuals. This means that the lowest levels of reported associations in the scientific literature likely and probably do not represent actual minimum threshold doses. It is unlikely that a true minimum exposure will ever be studied given ethics and safety concerns. However, with reasonable scientific certainty and based on sound scientific principles and methodology, we can detail levels of exposure to the toxins at issue that are hazardous to humans and are known to cause kidney cancer.

> There is an order of examination that is most appropriate in identifying low ranges of exposure associated with hazards to human health and that are known to cause kidney cancer. Given that the exposure of interest is water contaminated with multiple culprit compounds, the body of literature that directly examines the Camp Lejeune population exposed to the contaminated water system as measured by either duration of residence or the sum of these culprit compounds (TVOC) provides the most direct evidence for exposures at Camp Lejeune. Although the exposed group in this cohort is limited to those on base 1972-1985, Camp Lejeune exposures outside of this time window are similar in composition, although different in intensity, to the analyzed period with the primary exception of minimal PCE exposure prior to this period in the Hadnot Point water system (ATSDR PHA 2017).
>
> It is not likely that the majority of exposed were limited to a single water system on the base. However, TCE exposures dwarfed PCE exposures in the Hadnot Point water system, rendering such a difference in exposure composition largely irrelevant when using TVOC or duration as exposure metrics. Consequently, exposure levels associated with an increased risk of kidney cancer directly from the population of interest, with the exposure of interest, represent the best estimates of lower exposure levels hazardous to humans generally and known to cause kidney cancer.
>
> When a monotonic dose response is identified in this population, the lowest exposure metric with an elevated measure of association provides a conservative assessment of a lower exposure level hazardous to humans generally and known to cause kidney cancer. The true bound for equipoise is somewhere below this point, so the reported range is a conservative assessment of an exposure hazardous to human health taken directly from real world exposures. The presence of a monotonic dose response may allow for extrapolation to exposures outside of the studied population, providing an opportunity to extrapolate to exposures lower than the lowest exposure metric that exists.
>
> Given that the exposure of interest is water contaminated with multiple culprit compounds, the body of literature that directly examines the Camp Lejeune population exposed to the contaminated water system best answers the question of what levels of exposure are associated with kidney cancer.

Finally, it was made clear in the reports of Drs. Hatten and Bird that upper tract urothelial carcinoma should be analyzed under the umbrella of kidney cancer for purposes of assessing the epidemiological evidence in this case. As stated in the report of Dr. Hatten:

> The outcome of interest in this analysis consists of development of kidney cancers following exposure to the Camp Lejeune water system. Of note, some studies include urothelial/renal pelvis cancers with kidney cancers while some exclude them. Although more similar histologically to bladder tumors, most authors that do not separately analyze urothelial tumors include them with kidney cancers, and the measures of association in studies that include urothelial/renal pelvis cancers are similar to studies that do not include urothelial cancers (see appendix 1: table). Furthermore, in studies that directly compare urothelial/renal pelvis cancers to other kidney cancers, the measures of

association are similar (Lynge 1997; Raaschou-Nielsen 2003). Urothelial/renal pelvis cancers occur in the kidney. The kidney cancer epidemiological studies apply for purposes of this causation analysis. All four of the toxins at issue cause upper tract urothelial carcinoma.

I agree with these statements and the levels described in these reports. The reports were based on sound scientific principles and each of the reports cited well regarded and authoritative literature to support the opinions. I am relying on them, in part, for the analysis in this case.

Mr. Mousser meets and exceeds many of the levels shown to have been associated with increased kidney cancer incidence and mortality in the epidemiology studies taken directly from Camp Lejeune. As stated in the Bird and Hatten general causation reports, this is the most persuasive literature we have to assess causality in this case as it is the exact same mixture of chemicals, in the same amounts, as Mr. Mousser was exposed to at Camp Lejeune.

Mr. Mousser also meets many of the non-camp Lejeune levels cited above. The reason this is significant is because it shows the substantial and significant exposure for Mr. Mousser.

Mr. Mousser's exposure so far exceeds the levels shown in the literature to be causally associated with kidney cancer that his exposure to these toxins needs to be given great weight in a differential diagnosis analysis to determine the etiology of his kidney cancer.

IX. Differential diagnosis:

I have used a differential diagnosis methodology to determine the etiology of Mr. Mousser's kidney cancer. In order to do that, as stated above, I have made a list of all potential risk factors for Mr. Mousser's UTUC kidney cancer. The risk factors listed above are the medically valid and known risk factors for UTUC kidney cancer. To a reasonable degree of medical certainty there is sufficient evidence to conclude a causal relationship between Mr. Mousser's exposure to these toxins and the development of his renal/urothelial cancer. [redacted]